A STUDY OF VELOPHARYNGEAL CLOSURE IN CHILDREN WITH VOCAL NODULES

by

Bonnie Wagoner Amos

A Thesis Submitted to
the Faculty of the Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
of the Requirements for the Degree
Master of Arts

Greensboro
1972

Approved by

Mariana Newton

Thesis Adviser

AMOS, BONNIE WAGONER. A Study of Velopharyngeal Closure in Children with Vocal Nodules. (1972)
Directed by: Dr. Mariana Newton. Pp. 60.

The etiology of vocal nodules has eluded speech pathologists and physicians alike. The literature reporting incidence and etiology has been inconclusive. Few studies have suggested a physiological disorder as the etiology of vocal nodules. However, McWilliams, Bluestone, and Musgrave (1969), in noting the high frequency of vocal nodules in a population of cleft palate children, have suggested that velopharyngeal inadequacy may be a cause of vocal nodules in these children. The possibility that non-cleft palate children with vocal nodules also have minimal velopharyngeal inadequacy was proposed.

The method for data collection consisted of obtaining airflow measures on nine children with vocal nodules. These children ranged in age from eight to twelve years. In addition, an individual record was compiled on each child. This record contained information regarding the child's medical history, onset and development of hoarseness, variables affecting hoarseness, and history of vocal use. In addition, a space was provided on the record to note the occurrence of other speech disorders in the families of the subjects.

The results of the study revealed that all nine subjects had adequate velopharyngeal closure as measured by the airflow procedure. Therefore the hypothesis that these non-cleft palate children with vocal nodules also have velopharyngeal inadequacy must be rejected. The individual record results supported the literature in suggesting that vocal abuse, particularly during a time when the vocal cords are inflamed, is related etiologically to vocal nodules. The results on two subjects supported the theory that tension may be related to vocal abuse.

APPROVAL PAGE

This thesis has been approved by the following committee of the Graduate School at The University of North Carolina at Greensboro.

Thesis Adviser _____

Oral Examination Committee Members _____

_____

_____

February 11, 1972
Date of Examination

ACKNOWLEDGMENTS

The writer wishes to acknowledge with gratitude the extraordinary talent and patience of her thesis adviser, Dr. Mariana Newton. The writer further wishes to acknowledge the wisdom of the oral examination committee: Dr. Herman Middleton and Dr. Nancy White.

The writer is particularly grateful to Dr. Doris Bradley and to Dr. Donald Warren for their assistance with the research and for making the facilities at the Dental Research Center at Chapel Hill available for this study. Appreciation is extended to Dr. Bradley, who generously gave her counsel in the planning of the study, and to Dr. Warren, who directed the administration of the airflow measures and supervised the data analysis. A special thanks is due to Jeannine A. Mashburn, who conducted the hearing tests, and to Karen B. Barker, who assisted with the designing of the illustrations used in the text. Finally, the writer wishes to express gratitude to her family and friends for their encouragement and patience.

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CHAPTER I

INTRODUCTION

Vocal nodules in children are a problem for speech pathologists. Although the etiology of vocal nodules has not been specifically determined, much of the literature supports the theory that such nodules are the direct result of excessive vocal use. Therefore, vocal rest followed by voice training is the treatment which is generally recommended by speech pathologists. However, the usual play habits of children make vocal rest difficult, if not impossible, before or after surgical intervention.

Few studies have explored the possibility of a physiological factor in the etiology of vocal nodules in children. However, McWilliams, Bluestone, and Musgrave (1969) noted an unusually high incidence of vocal nodules in a population of children with congenital clefts of the palate. It was proposed that these children, with inadequate velopharyngeal closure, may attempt to use vocal cord valving activity to compensate for the poor closure. Thus, this compensatory valving movement could cause stress on the vocal cords and, therefore, could result in the development of vocal nodules. In view of the frequency of vocal nodules in children with palatal clefts, the possibility that vocal nodules in non-cleft palate children may be due to inadequate velopharyngeal closure is proposed.

Various techniques have been used in the past to determine velopharyngeal closure. For example, by looking in the mouth of the

subject, the closure can be indicated through observation of palatal elevation. Others have placed a mirror under the subject's nose while the subject was producing non-nasal speech sounds. Any clouding on the mirror revealed nasal emission of air and, thus, indicated poor velopharyngeal closure. Another technique has involved the use of a dry spirometer. The spirometer is designed to measure airflow through the mouth. Therefore, low measures of airflow through the mouth could be indicative of poor velopharyngeal closure. However, these techniques fail to provide a precise measure of the velopharyngeal orifice area. During the last decade, there have been significant improvements in the techniques for measuring vocal tract configurations, particularly velopharyngeal closure. For example, various X-ray techniques have been used to determine the degree of velopharyngeal inadequacy. Another method, the airflow procedure, was the technique selected for the purposes of this study. This procedure, which subtracts nasal pressure from oropharyngeal pressure, yields a precise measurement of small velopharyngeal gaps.

The purpose of this study was to investigate the extent of velopharyngeal closure in non-cleft palate children with a diagnosis of vocal nodules. It was hoped that such a study might provide some information of significance to the understanding of the precipitation and development of vocal nodules in children. Improved understanding of this disorder could lead to more effective clinical management of such cases and, possibly, to preventative measures.

CHAPTER II

REVIEW OF THE LITERATURE

The diagnosis of vocal nodules in children has often followed when hoarseness was the chief complaint. The medical literature has provided information about the nature of this condition; however, the investigations concerning the incidence and etiology of nodules have been few and inconclusive.

Definition of Vocal Nodules

Vocal nodules are described as growths which do not spread or effect general health but which may cause pain or hamper functioning (Brodnitz, 1953). Froschels and Jellinek (1941, p. 158) have stated that vocal nodules appear to be an ". . . epithelial alteration of the vocal cords in response to the irritation caused through exaggerated pressure of one cord against the other." More specifically, there are two major types of nodules: the reddish, soft, young nodule composed of normal squamous epithelium and the white, hard, mature nodule composed of thickened epithelium (Arnold, 1962). The development of nodules is continuous. Ash and Swartz (1944) have described four stages of epithelial alteration of nodules: fibroid, polypoid, varicose, and hyalin. The irritation of the young blood vessels extending toward the epithelium surface have characterized the fibroid state. Scattered growth of fibroblasts, connective tissue cells, have characterized the polypoid state. Varicose, the state often diagnosed as polypoid, is recognized by the presence of interstitial hyalin. Finally, the hyalin

state was referred to as the degeneration of connective tissue. Most authorities have agreed that vocal nodules are usually bilateral and occur at the point of greatest stress, which is generally on the anterior third of the cord (Arnold, 1962; Ash and Swartz, 1944; Harris, 1948; and Wilson, 1965). Although nodules are considered benign, they do hamper the functioning of the vocal cords, resulting in audible symptoms. Hoarseness is reported as the major voice symptom (Wilson, 1966). Physiologically, vocal nodules prevent adequate approximation of the vocal cords and interfere with the free vibration of the cords (Grey, England, and Mahoney, 1965). The resulting dysphonias may be classified as hoarseness and breathiness. In addition to these symptoms, low pitch, glottal plosive attack, narrow pitch range, and stress patterns may accompany vocal nodules (Berry and Eisenson, 1965). In summary, vocal nodules are considered to be benign lesions of the vocal cords resulting in hoarseness. These nodules are usually bilateral and generally appear on the anterior third of the cords.

Incidence of Vocal Nodules

The literature concerning the incidence of voice disorders in general has been sketchy; information about the incidence of vocal nodules is practically nonexistent. Johnson, et al. (1967) estimated that the incidence of voice problems in children is from one to two per cent of the population. Moore (1957) added that voice disorders occur in five to fifteen per cent of the defective speech population. In a survey to determine the number of speech handicapped persons in New England, Pronovost (1951) found that 6.6 per cent of a population of 12,565 had voice disorders. The data, collected by sending

questionnaires to those institutions offering speech and hearing services, failed to define voice disorders. A study to determine the need of a speech therapist in the Holyoke, Massachusetts schools indicated that 1.5 per cent of a population of 4,685 had voice disorders, with a greater incidence in grades one through three. An amusing aspect of this study was that the school system later employed the therapist who assisted in the initial screening for the survey (Mills and Streit, 1942). In a study of the incidence of chronic hoarseness in the Willow Run Public School System in Michigan, Baynes (1966) found an incidence of 7.1 per cent in a population of 1,012, with a greater incidence in the first grade. The procedure for this study involved three screening surveys at one month intervals. If the patient manifested hoarseness during all three surveys, the judges diagnosed the case as chronic hoarseness. Baynes, who excluded those cases of mild hoarseness, considered his results to be conservative.

Several studies have focused on the incidence of voice disorders in persons with congenital clefts of the palate. In a study of 1,061 clinic records on persons with cleft palate, Takagi, McGlone, and Millard (1965) reported the incidence of voice disorders other than nasality as .5 per cent of the males and .7 per cent of the females. Using 12 normal children as the control group and 76 children with cleft palate as the experimental group, Brooks and Shelton (1963) found that ten per cent of those with cleft palate have voice deviations such as hoarseness, breathiness, and inappropriate pitch. The between-judge reliability was .95. A possible explanation for the discrepancy in these two studies may be that one relied on clinic records, while the other resulted from experimental research.

McWilliams, Bluestone, and Musgrave (1969), studying minimal velopharyngeal inadequacy in children with congenital clefts of the palate, found that, in a population of 32 children with hoarse voices, 71 per cent had bilateral nodules. In addition, three children demonstrated left unilateral nodules, four demonstrated right unilateral nodules, and four had atypical vocal cords other than nodules. Included in these last four children was one child with edema of the vocal cords, who developed vocal nodules four months later.

In relating hoarseness to the occurrence of nodules, McCall (1970) states, "It has been my experience that 80-90 per cent of children with variable hoarse voices (i.e. degree of hoarseness varies) exhibit laryngeal pathology. The most frequent pathology observed is vocal nodules." In a study of 300 cases of benign vocal cord lesions, Fitz-Hugh, Smith and Chiong (1958) found 134 cases of nodules. In addition, of these 300 cases, 68.3 per cent occurred in males and 31.7 per cent occurred in females. No sex ratio was reported in relation to nodules specifically. Similarly, Arnold (1962) said that vocal nodules are the most common laryngeal lesion treated surgically; the occurrence is greater among males. However, others have reported a greater incidence of vocal nodules in females. In a study of 1,160 cases of functional voice disorders, Garde (1961) found 97 cases of unilateral nodules and 168 cases of bilateral nodules. Included in the 97 cases of unilateral nodules, were 92 women, 4 men, and 1 child. Among the 168 cases of bilateral nodules were 164 women, 3 children, and 1 man. Perhaps the greater incidence among women may be explained in part by the fact that 93 of the cases studied were school teachers. Although no specific

numbers are reported, Zerffi (1935) reported a greater incidence of nodules among females. Zerffi based his observations on singers. In summary, although none of these findings were conclusive, the information to date appeared to concur with the opinion that the incidence of vocal nodules is relatively infrequent among children (Wilson, 1965).

Etiology of Vocal Nodules

In turning to the causes of vocal nodules and related voice disorders, the literature appeared repetitive and scarce. Van Riper and Irwin (1958, p. 279) have offered a possible reason.

> The literature is scanty and scattered. Except for certain occupations such as the ministry, teaching, and entertaining, the average voice defect is not a handicap, since communication is still possible, a factor which does not hold true in stuttering or articulatory defects.

However, the literature concerning the causes of vocal nodules and related voice disorders does lend itself to grouping into four major categories: vocal abuse, physical causes, psychological causes, and mixed causes.

Some authorities have expressed the opinion that vocal nodules are the result of vocal abuse. Greene (1957, p. 78) has written, "The nodules which form on the outer edge of the cords and cause severe dysphonia are the direct outcome of vocal abuse and the individual's habitual method of forcing the voice." In referring specifically to vocal nodules in children, Arnold (1962, p. 214) has said, "In screaming children, bilateral vocal nodules result from and demonstrate the presence of excessive and uncontrolled vocal expression." To clarify this point, Arnold (1962, p. 214) stated that "In children, nodules result from excessive yelling, singing, or vociferous outdoor play."

Furthermore, in a study of 138 persons with vocal nodules, Ash and Swartz (1944) found that all but 9 cases were due to excessive, loud talking as determined by case history reports. In this study, a specific instance of vocal abuse was the vocal habits of the drill sergeant. Seventy-five of the 138 persons were Army personnel.

However, there are those (West, et al., 1947, pp. 140-41) who have said that "No amount of vigorous vocalization can damage the edges of the vocal folds if the voice is properly used." Zerffi (1935, pp. 552-53), in discussing singer's nodules, related the improper physiological production of high pitches to a theoretical cause of singer's nodules.

> The action of the arytenoid and lateral cricoarytenoids, and the thyroarytenoids is that of bringing the posterior edges of the cords closely together and thus shortening the vibrating length. This approximation helps to raise the pitch of the tone, and high tones are thus sung with the anterior two-thirds or even with the anterior half of the cords. Since nodules occur at the junction of the anterior third of the cords, high tones sung as described could not possibly produce an irritation of the anterior third. Nor are there any laryngeal muscles which act in such a manner as to provide sufficient pressure at this point to result in the formation of nodules. It is therefore obvious that other muscles than those of the larynx proper are concerned in this action. In the opinion of this writer, the muscles which bring about this pressure are those which are concerned in deglutition. These muscles, notably those of the tongue, the action of which can be detected by means of finger palpation, assist those of the larynx in bringing about complete closure of the larynx when deglutition is about to be performed. Forced production of the voice is brought about by a similar action as that employed by deglutition. A partial contraction of the muscles is used to force the vocal cords into approximation and only by generation of considerable energy can the breath be driven through the glottis.

McWilliams, Bluestone, and Musgrave (1969, p. 3), in a study of 32 children with hoarse voices and congenital clefts of the palate, found that 84 per cent demonstrated pathology of the vocal cords. Of this 84 per cent, 59 per cent had borderline velopharyngeal inadequacy

as determined by radiographic tapes. Therefore, these writers suggested:

> . . . inadequate velarpharyngeal valving mechanisms might be related to vocal cord nodules in a logical if not a readily demonstrable manner. This idea was reinforced by information to the effect that 16 of the 22 children with vocal cord nodules had had speech therapy prior to their laryngeal diagnoses. It appeared to us that compensatory valving activities, even in the absence of glottal fricatives and plosives, might be one means by which children would attempt to handle problems high in the tract thus subjecting the vocal cords to stress.

An interesting aspect of this study was that one child developed nodules following therapy sessions designed to evaluate his velopharyngeal closure (McWilliams, Bluestone, and Musgrave, 1969, p. 4).

A third group of studies have concentrated on the psychological causes of nodules and associated voice disorders. In this group is a study of reciprocal inhibition as a treatment for nodules. In describing a method of treatment Grey, England, and Mahoney (1965, p. 188) wrote:

> The basic premise of the present therapy is that there are certain people in whom benign functional vocal nodules develop as a result of pervasive anxiety. This type of patient is seen as a person who is in a more or less constant state of anxiety or stress. This anxiety is manifested in emotional over-reaction to situations and misuse of the vocal apparatus due to psychological and physiological stress. Originally, isolated situations may have been responsible for high anxiety states--the anxiety reaction being an unconditioned response to the stimulus situation. However, as more and more cues became associated with the stimulus situation the anxiety reaction became a conditioned reaction to a variety of conditioned stimuli which formerly did not evoke anxiety--the result being pervasive or free floating anxiety. Thus, the patient is seen as moving from isolated situations of anxiety to a more or less constant state of anxiety.

This report implied the opinion that vocal nodules appear to be related etiologically to anxiety states. The authors have suggested a treatment program which includes the patient's acknowledgment of feared

situations, followed by practiced non-anxiety reactions to these situations (Grey, England, and Mahoney, 1965).

Although Aronson, Peterson, and Litin have dealt primarily with voice disorders in adults which are due to psychological factors, their research was interesting to note. In one study (Aronson, Peterson, and Litin, 1964), the authors classified vocal nodules as a direct result of misuse and described voice disorders, such as spastic dysphonias and ventricular dysphonias, as being characteristic of psychoneurotic or psychotic mental states. These disorders manifest audible symptoms similar to those of vocal nodules. In 1966 Aronson, Peterson, and Litin administered the Minnesota Multiphasic Personality Inventory to 27 persons with voice disorders (spastic and ventricular dysphonias). These persons ranged in age from 14 to 72. As a result of this study, Aronson, Peterson, and Litin (1966, p. 126) concluded that "Acute or chronic situational conflicts were causally related to the voice disorders in the overwhelming majority of patients, regardless of type of voice symptomatology." Furthermore, Alfaro (1960) reported that voice disorders due to psychogenic influences were more common in adults than in children. Contrary to these studies, Goodstein (1958, p. 364), in a review of psychological causes of voice disorders, concluded that ". . . the relationship between voice disorders and personality is yet to be empirically demonstrated."

By far, the bulk of literature has concentrated on a combination of factors as causing vocal nodules and associated voice disorders. That is, when speaking of vocal abuse, physical factors, and psychological factors as causes of vocal nodules, some cases may be due to a single cause, but the majority have been the result of a combination of

all three (Withers, 1961). Several writers have discussed these three causal factors while others consider only two factors to be interacting.

In discussing all three aspects, Brodnitz (1958, p. 112) stated that the fact that nodules occur on the vocal cords, ". . . does not mean that faulty vocal cord function alone is to be blamed." Brodnitz (1958) listed eight factors to consider: the breathing mechanism, the resonation cavities, mutational changes, tenseness during speech, excessive talking or shouting, faulty voice production during singing, daily emotional tenseness, and hormonal or metabolic imbalance.

Froschels and Jellinek (1941, p. 194) discussed the role of vocal abuse.

> . . . . Professional singers and speakers such as teachers, politicians, lecturers, lawyers, preachers, etc. who have to stand great professional strain without sufficient technical preparation often try to overcome fatigue or decrease of their vocal capacities through some acute disease, for instance, a cold by augmented use of force. They tighten muscles which should work with ease. The effect is an ever increasing overstrain of these muscles, which may even result in some organic alteration, such as bursting of small vessels, irritating sensations with resulting coughing. The nodules of the vocal cords, so greatly feared by singers, are among the effects of such overstrain.

In describing types of physical hyperfunctions, Froschels and Jellinek (1941) listed six hyperfunctions: violent forcing of air through the vocal cords, 'coup de glotte,' contraction of pharyngeal muscles, retraction of the tongue, excessive elevation of the velum, and tenseness of the lips. Moreover, a study of 1000 persons revealed that contraction of pharyngeal muscles and 'coup de glotte' were the factors which occurred most often in children speaking under stressful situations. In addition, Froschels and Jellinek (1941, p. 195) offered the following consideration of the psychological factor:

> The psychic phenomena accompaning every decay of the vocal qualities in a singer are striking, and greatly increase his difficulties. These difficulties in singing are usually far greater than we should expect after examination of the vocal cords. They originate in part from the exaggerated attention which the patient--singer or professional speaker--directs toward his own phonetic function.

Besides faulty usage, Arnold (1962, pp. 205-6) cited three additional causative factors: 'predisposing,' 'precipitating,' and 'aggravating.' Included in the 'predisposing' factors were persons with aggressive personality structure, persons inclined to allergies, and persons with poorly constructed vocal mechanisms. The 'precipitating' factors were tobacco and alcohol, accompanied by vocal abuse. That is, during social gatherings, alcohol may promote mucosal hyperemia (a concentration of blood cells) of the vocal cords, and then tobacco may irritate the vocal cords. This irritation, resulting in hoarseness, could cause hyperkinetic efforts to talk louder.

Wilson's discussions of vocal nodules and laryngeal dysfunction were of interest. Regarding vocal nodules in children, Wilson (1961) listed seven causitive factors: inappropriate pitch, excessive air force during phonation, excessive talking, loud phonation, abrupt phonation, excessive strained phonation during play, and emotional factors. Later Wilson (1966, pp. 76-78) listed the following suggested goals of voice therapy which explained the above factors in more detail and mentioned the additional factors of posture and rate of speaking.

> 1. Vocal Abuse
> Attention must be given to vocal abuse in all benign voice pathology patients. Abusive habits include the frequent use of such traumatizing vocal practices as vigorous throat clearing, excessive and hard coughing, vocalizing on intake of air, screaming and shouting, prolonged vigorous use of the voice and excessive talking, and emitting strained vocalizations. . . .
> . . . . . .

> 2. Easy Initiation of Tones
> Patients with benign vocal pathology may speak with sudden, abrupt initiation of sounds resulting in an irregular and staccato type of speaking. . . . This practice, when forceful and traumatizing, may result in nodules or polyps on the vocal cords, contact ulcers on the vocal processes or the arytenoids, or nonspecific laryngitis.
> . . . . . .
> 3. Desirable Pitch Change
> . . . . That is, some patients may use an abnormally high pitch when they speak loudly under noisy conditions, talk under emotional strain, or give a public speech. Other patients may use an abnormally low pitch in certain situations, such as when talking on the telephone, giving a sales pitch, or participating in small group discussions and conferences. . . .
> . . . . . .
> 4. Appropriate Loudness of the Voice
> The habitual speech of some patients is so loud that irritation of the vocal mechanism results. . . .
> . . . . . .
> 5. Relaxation, Correct Breathing Patterns, and Good Posture
> . . . . Specific relaxation is especially necessary when undue tension of certain laryngeal muscles is contributing to the pathology or when continued overuse has led to hypofunction and weakness. Efficient breathing patterns and good posture may also need attention.
> . . . . . .
> 6. Rate of Speaking
> Any deviations from a desirable rate of speaking should be corrected. An excessively rapid rate of speaking is characteristic of many patients with vocal pathology and may indicate faulty use of the vocal mechanism. . . .

Rather than emphasizing the psychological aspect, others concentrated more on the vocal abuse and medical or physical aspects. For example, Berry and Eisenson (1956) reported vocal abuse as the probable cause of vocal nodules in 90 per cent of the cases. Included as types of vocal abuse were loud phonation and inappropriate, low pitch. Continuing, Berry and Eisenson (1956, p. 212) gave attention to the aspect of tension in the following statement.

> The effect of the tension, whether external or internal, is to restrict the freedom of movement, particularly of the anterior one-third section of the vocal folds. As a result of tension and contact, the area becomes degraded, and increased layers of epithelium are built up forming the nodule.

West, Kennedy, and Carr (1947) cited that repeated misuse of the vocal mechanism and chronic laryngitis or tonsillitis can lead to nodules. Similarly, Alfaro (1960, p. 6) said that "Abnormalities of phonation in the child are usually on an organic basis such as vocal nodules, from misuse of the voice, or laryngitis of either infectious or allergic etiology."

In clarifying the meaning of vocal abuse, Van Riper and Irwin (1958) mentioned vocal abuse in its relationship to physical factors. That is, vocal abuse may include excessive use of the voice, particularly if the vocal cords were inflamed, speaking 'on residual air,' speaking loudly over a continuous masking noise, speaking with inappropriate pitch and loudness, and hypertension of the laryngeal muscles used in swallowing while speaking.

Placing less emphasis on the physical aspect, Rubin and Lehrhoff (1962, p. 153) saw the causes of vocal nodules as being ". . . an extended spectrum ranging from simple loud phonating at one end to unadulterated emotional tension at the other, with varying combinations of the two between." In clarifying the importance of vocal abuse as a cause, Rubin and Lehrhoff stress the relativity of vocal abuse to individual speakers. That is, what constitutes vocal abuse for one speaker might not necessarily be detrimental to another speaker (Rubin and Lehrhoff, 1963).

In summary, vocal nodules are defined as benign lesions of the vocal cords resulting in hoarseness. Although the reports were inconclusive, the information indicated a relatively infrequent incidence of nodules in children. Concerning etiology, the literature has concentrated on four types: vocal abuse, physical causes, psychological

causes, and mixed causes. The majority of the literature emphasized the interaction of several factors to produce vocal nodules.

CHAPTER III

PROCEDURES

The etiology of vocal nodules has eluded speech pathologists and physicians alike. The etiological studies have been few and inconclusive. Most of these studies conclude that nodules result from loud, excessive vocal use. Few studies investigate the possibility of a physiological inadequacy in relation to vocal nodules. However, McWilliams, Bluestone, and Musgrave (1969) have reported that children with inadequate velopharyngeal closure, related to congenital clefts of the palate, frequently develop nodules. Therefore this study was designed to investigate velopharyngeal adequacy, not related to palatal clefts, in children with vocal nodules. The subjects, the instrumentation, and the procedure will be discussed below.

Subjects

The nine subjects used in this study consisted of six males and three females. Six of the subjects were selected from the client records at the University of North Carolina at Greensboro Speech and Hearing Center. The remaining three subjects were referrals from the Guilford County Health Department, a practicing laryngologist in Greensboro, and the Dental Research Center at Chapel Hill, North Carolina.

With regard to the previous treatment of the subjects at the time of this study, three of the subjects had had voice therapy following the surgical removal of the nodules. The remaining subjects reportedly had had voice therapy which concentrated on general

relaxation, alterations in vocal pitch, and a decrease in vocal abuse. No therapy had been centered on velopharyngeal adequacy or nasality problems.

The initial contact with the parents of the subjects involved sending a Letter of Introduction. (See Appendix I, page 57). The purpose of the letter was to provide the parents with a general concept regarding the nature of the study. After the letter, each parent was contacted by telephone. This telephone contact served to answer any questions the parents had regarding the study and to further solicit their cooperation.

Prior to being selected as a subject for the study, a confirmation of the diagnosis of vocal nodules in each subject was needed. Clinic records revealed that each of the subjects had had a laryngeal examination by practicing laryngologists in Greensboro or Chapel Hill. These records showed that the three female subjects and five of the male subjects had bilateral nodules. One male subject had a unilateral nodule.

The subjects were limited to those children between the ages of eight and twelve years. The upper age limit was specified hopefully to exclude those children experiencing voice mutation. However, due to the physical appearance and voice quality of one twelve year old child, it was decided that this child's voice was possibly undergoing mutation. Therefore, to avoid contamination of the data, this child was excluded from the study.

In addition, the parents were required to sign a Statement of Informed Consent. (See Appendix II, page 58). This statement explained the requirements for participation in the study. These requirements

stated that the parents were to supply information for an individual record and that each child was to have hearing and airflow evaluations.

The Individual Record was designed to collect information concerning the child's medical history, the onset and development of hoarseness, and a history of the child's vocal use. Also, a section was included to note the occurrence of other speech disorders in the subject's family. (See Appendix III, pages 59-60).

The literature cites inappropriate use of the vocal mechanism as a possible cause of vocal nodules. Since faulty voice production can occur in those persons with a hearing loss, a pure-tone audiometric sweep test was administered in a sound proof room to each subject. The procedure for the sweep test, suggested by Newby (1964), was used.

The parents were informed that their children would be taken to the Dental Research Center in Chapel Hill for the airflow evaluation. Transportation to and from Chapel Hill was provided as a convenience to the parents.

Testing Procedures

Individual Record

The literature has cited upper respiratory infection and vocal abuse as possible causes of vocal nodules. Therefore, to supplement the data, an individual record was kept on each subject. This record included questions regarding: (1) the child's medical history; (2) description and management of the child's vocal nodules; and (3) the development of the child's voice problem. Additional sections of the record provided spaces for identification information and the

results of the hearing test. Information for the Individual Record was acquired in a parent interview conducted by a graduate clinician.

Equipment

The equipment used for the study included a tape recorder, microphone, differential pressure transducer, heated pneumotachograph, and a Honeywell Visicorder and analogue computer arranged as shown in Figure 1, page 26. This equipment recorded velopharyngeal orifice differential pressure, volume rate of airflow through the orifice, and computed velopharyngeal area according to the formula given below. Speech was recorded through the use of a microphone placed under the subject's chin. Thus, speech appeared on the visicorder printout and helped to identify points on the record that should be measured. A tape recorder was placed approximately three feet from the subject so that a record could be made of the test phrases. This record served to verify, during analysis, the accurate order of presentation.

Velopharyngeal orifice differential pressure was determined through the use of two catheters, one in the subject's mouth and one in the nose, leading to the differential pressure transducer. So that the catheters would pick up only lateral pressure, both catheters were plugged at the end with wax and small holes were drilled along the sides of the catheters. Utilizing these two measures, the transducer subtracted the nasal pressure from oropharyngeal pressure to yield the velopharyngeal orifice differential pressure (Warren, 1964). The transducer then directed this electrical information into the computer.

The aspect of velopharyngeal airflow was detected by a flowmeter attached to plastic tubing placed in the subject's right nostril

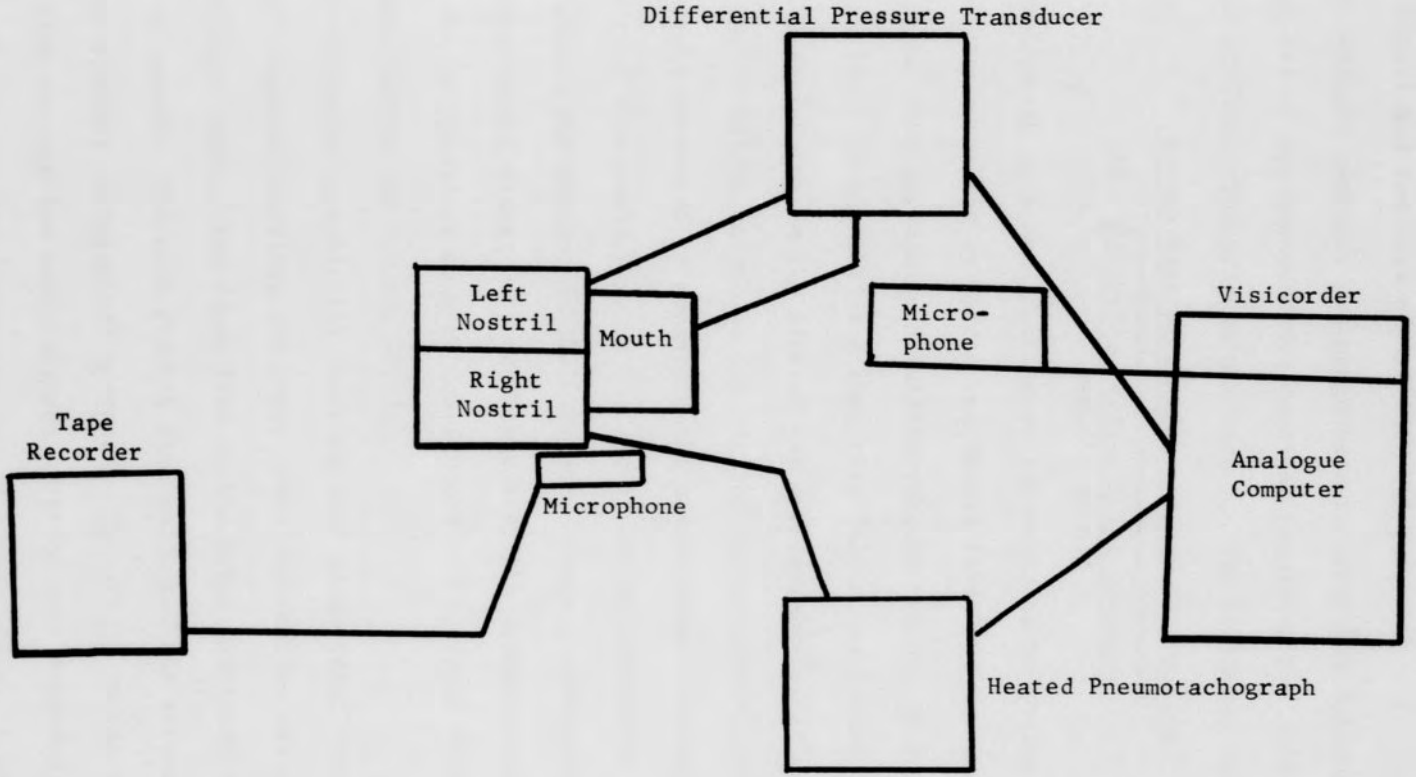

Fig. 1. Schematic Representation of the Equipment

(Warren, 1964). A transducer changed this flow measurement into an electrical signal and fed this signal into the computer.

The analogue computer was programmed to determine velopharyngeal area through use of the measurements received regarding the differential pressure and airflow. The program was based on the following equation:

$$\text{Area} = \frac{\text{Volume rate of airflow through the orifice}}{.65 \sqrt{2\left(\frac{\text{Orifice differential pressure}}{\text{Density of Air}}\right)}}$$

This equation, which is a modification of one reported by Gorlin and Gorlin (1951), was adapted by Warren and Dubois (1964) to measure velopharyngeal area. The formula was modified through the use of a correction factor (.65). It was believed that this factor was needed to account for the variations in the size of the oropharyngeal orifice due in part to the turbulent nature of the airflow during speech. Sixteen experiments were conducted to ascertain the effect which alterations in oropharyngeal size would have on the amount of the correction factor needed. However, the changes in the correction factor, resulting from various oropharyngeal sizes, were so slight that the decision was made to compute the arithmetical mean of the factors and to treat this mean as a constant (Warren and Dubois, 1964).

The visicorder graphically recorded four parameters: speech, differential pressure, airflow, and area. This information was printed on photosensitive paper. The first line of the graph indicated the occurrence of speech. The next graphic plot reflected the alterations in the velopharyngeal differential pressure. Variations in the volume rate of airflow through the velopharyngeal orifice were recorded as the third plot. The fourth line indicated velopharyngeal area. A schematic diagram of the printout appears in Figure 2, page 28.

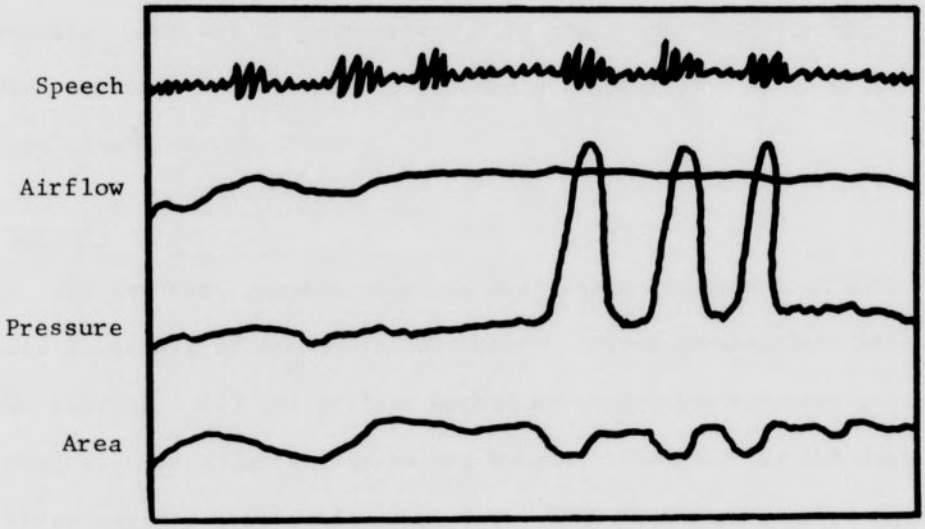

Fig. 2. Schematic Diagram of the Printout

Calibration

The apparatus was calibrated following the evaluation of each subject. The calibration procedure consisted of attaching the two catheters to a model of a velopharyngeal orifice and the flowmeter to a model "nose." The models were constructed of plastic tubings and joints. The simulated airflow of speech was furnished by an air pump. This cylinder introduced a known quantity of air into the model. The differential pressure transducer and flow transducer fed electrical impulses into the computer which yielded a graphic plot of an area equal to $.5cm^2$.

Test Phrases

The ten test phrases selected were short and redundant but included a variety of occlusive consonants. These phrases were selected for two reasons: (1) the airflow method of evaluation was most reliable with occlusive consonants (Warren and Dubois, 1964) and (2) the data from these phrases were easily analyzed. These ten phrases were presented aloud to each subject, and the subject was asked to repeat the phrase exactly as he heard it. The phrases were as follows:

1. say boo boo
2. say bee bee
3. say bah bah
4. say bay bay
5. say boh boh
6. say poo poo
7. say pee pee
8. say pah pah
9. say pay pay
10. say poh poh

Airflow Procedure

Each subject was seated on a stool in front of the table where the transducers were affixed. The height of the stool was adjustable so that the child could be positioned in a manner suitable for the insertion of the catheters and tubing. The catheters and tubing were inserted as described above. Each subject was instructed to say sample test phrases in order to determine if the tubes were properly positioned and to familiarize the child with the test procedure. One problem associated with the procedure existed in the fact that the children were reluctant to use their usual manner of articulation while the catheter was in place. It should be noted that erratic printouts from the visicorder were usually indicative of the presence of fluid in the catheter. In this event, the catheter had to be cleaned before the evaluation could continue. In addition, deflection of the catheter by the subject usually resulted in erratic printouts. These printouts did not provide an accurate measure of velopharyngeal area.

Three persons participated in the procedure: (1) one to hold the catheter and tubing in the subject's nose; (2) one to present the test phrases to the subject; and (3) one to manage and monitor the computer. The tape recorder was turned on prior to the evaluation of each subject. (See Figure 3, page 31).

Each child was instructed to repeat the test phrases one at a time. With three syllables per test phrase, there resulted thirty velopharyngeal orifice area measurements for each subject. One subject was found to have an articulation disorder; he distorted occlusive consonants. Therefore, two trials of the test phrases were recorded to insure adequate data.

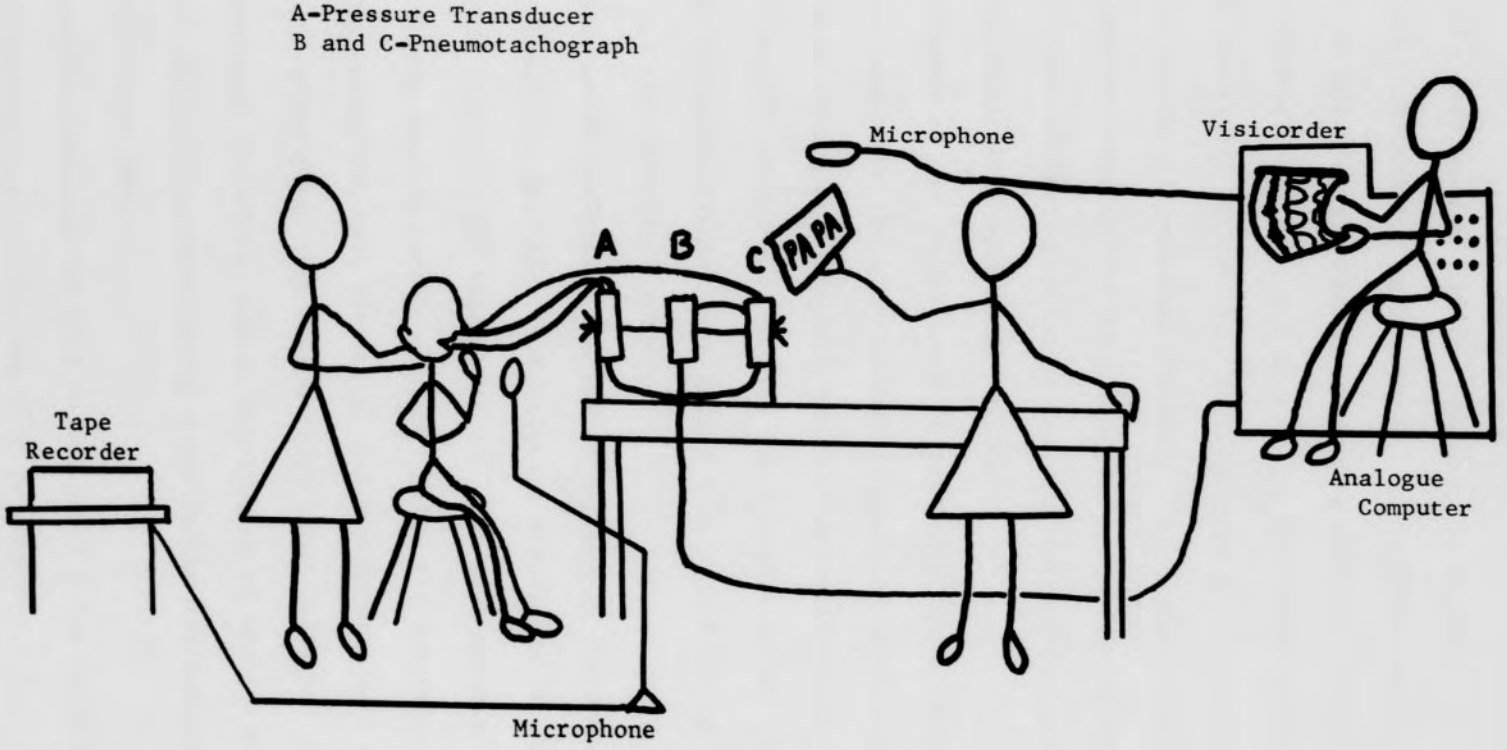

Fig. 3. Schematic Representation of Procedure Used in Recording Airflow Data

Data Analysis

The data consisted of airflow measures on the ten test phrases and calibration printouts for each subject. The airflow data were in the form of a graphic representation of four parameters: speech, differential pressure, airflow, and area. The first line of the graph indicated the occurrence of speech. The next graphic plot reflected the alterations in the differential pressure. Variations in the volume rate of airflow were recorded as the third plot. The fourth line indicated area. The calibration printout for each subject indicated a baseline and a linear area equivalent to .5cm^2.

The purpose of the analysis was to determine the velopharyngeal closure during speech for the nine subjects. Analysis of the airflow data consisted of recording the speech samples appropriately on the graphic plot from the visicorder, marking the baseline on the graph, and computing the velopharyngeal area during each repetition of the ten test phrases for the individual subjects.

The purpose in indicating the speech samples at appropriate points on the graph was twofold: to facilitate discussion of the data and to assist in comparing the velopharyngeal area measurements in the same subject during varying speech samples. While playing the tape recording taken during the test situation, the appropriate test phrases were written above the first line on the graph which indicated that speech had occurred. Therefore, casual examination of the graph revealed the test phrase being used during a particular measurement of velopharyngeal orifice area.

The baseline revealed the area on the graph below which the velopharyngeal orifice area was too small to be computed. That is,

when the line indicating velopharyngeal area fell below this baseline, the orifice area could be regarded as adequate. (See Figure 4, page 34). Furthermore, when the area line appeared above the baseline, the velopharyngeal area could be computed to determine its adequacy. (See Figure 5, page 34). Since this baseline appeared only on the calibration sheet for each subject, the line had to be drawn in manually on each graph.

The calibration showed a graphic plot of an area equal to .5cm^2. (See Figure 6, page 34). Therefore, in order to compute the velopharyngeal orifice area, a metric ruler was used to make two measurements: the linear equivalent to .5cm^2 and the distance in centimeters between the baseline and the area line. This second measurement was made only on the sections of the baseline and area line which appeared directly below peaks in the orifice differential pressure line. That is, as differential pressure increased, orifice area decreased. Furthermore, this second measurement was required only if the orifice area line appeared above the baseline. No measurement was necessary when the orifice area line fell below the baseline. To compute the orifice area, the two measurements were used in the following simple relationship equation:

$$\frac{\text{.5cm}^2 \text{ or } 50\text{mm}^2}{\text{The linear measurement on the calibration sheet equal to .5cm}^2} = \frac{\text{Velopharyngeal orifice area}}{\text{The distance in centimeters between the baseline and the area line}}$$

The results to the equation were expressed in square millimeters. Using subject B. M. as an example, if the linear equivalent to .5cm^2 were 5.2cm and if the distance between the baseline and the area line were .2cm, the area would be computed as follows:

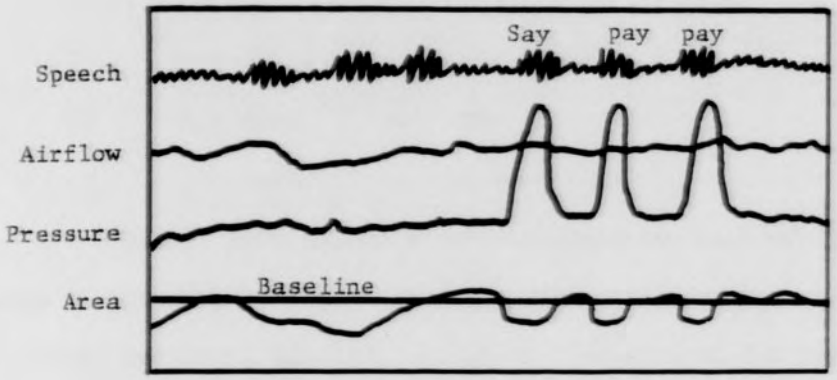

Fig. 4. Airflow Data Indicating a Velopharyngeal Area Too Small to be Computed.

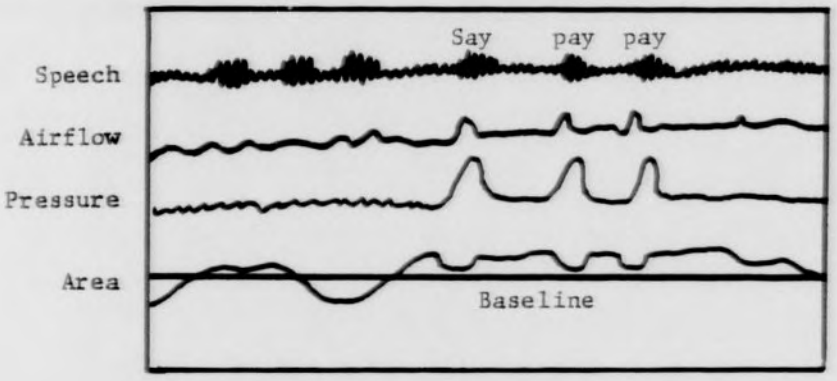

Fig. 5. Airflow Data Indicating an Adequate and Yet Measureable Velopharyngeal Orifice Area

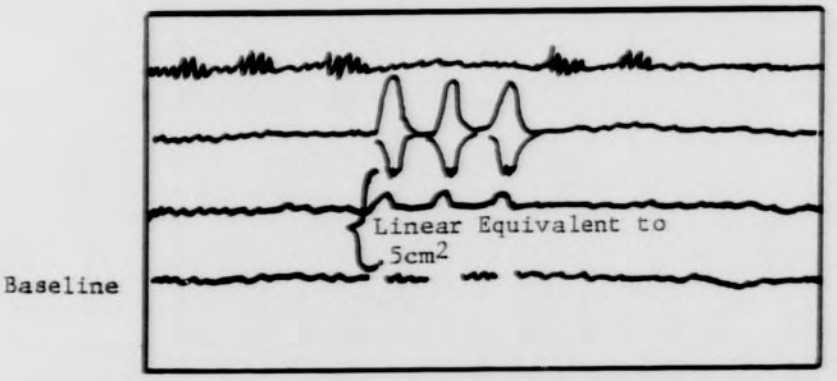

Fig. 6. Calibration Data Indicating the Baseline and an Area Equivalent to .5cm^2

$$\frac{50mm^2}{5.2cm} = \frac{Area}{.2cm}$$

$$(5.2)(Area) = (.2)(50)$$

$$5.2\ Area = 10$$

$$Area = 1.9mm^2$$

In summary, five measures were obtained for each of the nine subjects in this study. Four were recorded by the computer: speech, differential pressure, airflow, and area. The area of the velopharyngeal orifice, was calculated using the procedure described above. An analysis and interpretation of the findings is presented in the following chapter.

CHAPTER IV

RESULTS AND DISCUSSION

Data on nine subjects with vocal nodules were obtained in order to determine if these subjects manifested minimally inadequate velopharyngeal closure during speech. The primary method of data collection consisted of airflow measures on each subject while repeating ten phrases. In addition, an individual record was kept on each subject. With parents serving as informants, information was recorded regarding each subject's medical history, the onset and development of hoarseness, the variables affecting hoarseness, and the occurrence of other speech disorders in the families of the subjects. These measures provided the data which are presented and discussed in this chapter.

Velopharyngeal Area Results

A summary of the velopharyngeal area measurements on each subject is presented in Table 1, page 38. As is indicated on this table, 27 measurements out of a possible 252 instances were calculated. These 27 measurements were conducted on six of the subjects. Three subjects, B. M., B. C., and T. S., each had one syllable out of the ten test phrases during which their velopharyngeal area could be computed. The results, expressed in square millimeters, were (Say) 4.3, (Say) 1.1, and (Bee) 1.9. The results on D. L. indicated a measurable velopharyngeal area on two syllables out of the ten test phrases. These syllables were (Bay) 3.1 and (Pee) 7.8. Three velopharyngeal area measurements, (Bee) 4.7, (Say) 4.7, and (Poh) 7.1, were computed on T. M.

Interestingly, one subject, R. C., had measurable orifice areas ranging in size from 2.2 to 1.2mm^2 on nineteen syllables. However, due to the fact that this subject had an articulation disorder which involved poor production of the consonants in the test phrases, caution should be exercised in interpreting his area results. That is, his manner of articulation may have affected his velopharyngeal port area. Nonetheless, the airflow results on these 27 measurements failed to approach the established critical level of 20mm^2 (Warren, 1964) for inadequate velopharyngeal closure.

No calculations were necessary or could even be conducted on the remaining 225 syllables because the graphic printout from the computer showed the area line below the baseline. That is, the area was too small to be computed and, therefore, was regarded as adequate. In fact three subjects, J. H., M. C., and B. F., required no area calculations at all. The other six subjects had a combined total of 117 syllables on which no calculations were necessary.

In summary, 27 velopharyngeal measurements out of a possible 252 could be conducted on six subjects. These measurements ranged in size from 1.1mm^2 to 12.2mm^2, with a mean of 5.5mm^2 and a standard deviation of 2.59mm^2. None of the measurements approached the significant velopharyngeal port size of 20mm^2. Such an area would have been indicative of inadequate velopharyngeal closure.

Individual Record Results

The individual records were forms on which information regarding each subject's medical history, the onset and development of hoarseness, the variables affecting hoarseness, and a history of vocal use was

TABLE 1

AREA MEASUREMENTS, REPORTED IN SQUARE MILLIMETERS,
ON NINE SUBJECTS SAYING TEN PHRASES

		Subjects								
		R.C.	T.M.	D.L.	T.S.	B.C.	J.H.	M.C.	B.F.	B.M.
Test Phrases	Say	3.3	-			1.1				
	Boo	3.3	-							
	Boo		-							
	Say	12.2								1.9
	Bee	5.6								
	Bee	4.7	4.7							
	Say	7.3				x				
	Bah	6.7	-			x				
	Bah	5.6	-			x				
	Say	-								
	Bay			3.1						
	Bay	2.2								
	Say	3.3			4.3					
	Boh	5.6								
	Boh	7.8								
	Say	2.2								
	Poo									
	Poo									
	Say	7.8	-							
	Pee		-	7.8						
	Pee		-							
	Say	10.0								
	Pah	7.8								
	Pah	5.6								
	Say	-	4.7							
	Pay									
	Pay									
	Say	7.8	-							-
	Poh		7.1							-
	Poh									-

(-) Indicates deflection of the catheter resulting in no measure.
(x) Indicates that the phrase was not presented to the subject.

recorded. In addition, questions were included regarding etiology and the occurrence of other speech disorders in the families of the subjects. The results of these individual records are presented below.

Upper Respiratory Infections

It was interesting to note that of the nine subjects used in this study, eight reportedly had a history of upper respiratory infections. Included in these eight were four subjects whose respiratory infections were said to be unrelated to the onset of hoarseness. For example, the individual records revealed that R. C. had frequent sore throats and tonsillitis. J. H. had chronic colds as an infant and an allergy to pollen. In addition, he had an adenoidectomy at the age of six. T. S. had symptoms of pneumonia at the age of five. B. M. was said to suffer from allergies during the Spring of each year.

The remaining four subjects reportedly had upper respiratory infections at the time when hoarseness first became noticeable. Included in these four were B. F., D. L., T. M., and M. C. B. F. had tonsillitis at the age of five and a tonsillectomy at the age of six. Then, between the ages of eight and nine, the age of the onset of hoarseness, she had chronic sore throats and colds. D. L. had frequent mild sore throats as an infant and again between the ages of five and six. The age of five was the approximate age of the onset of hoarseness. T. M. had chronic colds and laryngitis during the period when hoarseness began. M. C. had frequent sore throats at the age of eight, the age when hoarseness first became noticeable. Therefore, of the nine subjects under study, eight subjects or 88 per cent of the sample had a history of upper respiratory infections. Four of the subjects reportedly had

respiratory infections which appeared to be related to the onset of hoarseness.

Medication

Three of the subjects, who had experienced upper respiratory infections, had taken certain prescribed medications. For example, B. F. took Dimetapp during her frequent attacks of sore throats and colds. J. H. reportedly had been on Triaminic for his allergy to pollen. Also, this subject has been taking Dexedrine for hyperactivity. The third subject, B. M., has taken Histalet to control conjestion due to allergy. In summary, both subjects who were said to have allergies were taking a prescribed medication. However, only one of the subjects who had had chronic colds and sore throats had taken a prescribed medication to control such infections.

Age of the Onset of Hoarseness

When the mothers of each subject were questioned regarding the age of the onset of hoarseness, four mothers placed the onset at age eight, one at age nine, and one at age ten. R. C., serving as his own informant,[1] and the two remaining mothers identified the onset at much younger ages. R. C., who developed a hoarse voice at the age of three, had the earliest reported age of onset. D. L.'s hoarseness became noticeable at the age of five. B. C.'s mother recalled that the

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[1]R. C. was recently placed in a foster home for reasons not specified to the examiner. However, clinic records and information acquired from the staff at the Dental Research Center in Chapel Hill assisted in assuring the reliability of the information received from R. C.

hoarseness developed at the age of six. Therefore, the ages of onset ranged from three to ten years. However, six subjects reported the onset to be between the ages of eight and ten years.

Development of Hoarseness

When asked to describe the course of the development of their children's hoarseness, six mothers recalled that the hoarseness gradually progressed from slight to more severe. Two subjects had hoarseness which was described as being constant in severity since the initial occurrence. M. C.'s mother reported that the severity of the hoarseness has varied from time to time. Therefore, although three subjects had hoarseness which was described as either constant or varying, six of the subjects had hoarseness which was judged as following a gradual developmental course.

Variables Affecting Hoarseness

Except for R. C., whose hoarseness reportedly was at a constant state at all times, the remaining subjects listed a number of variables which negatively affected the hoarseness. The most common variables reported were prolonged talking and vocal abuse. These factors were relative to seven subjects. The individual records of four subjects indicated additional variables. For example, B. F.'s hoarseness worsened during periods of tension. T. S., T. M., and D. L.'s hoarseness became more noticeable after they had been singing. B. M.'s hoarseness was judged by her mother as being more severe in the morning and evening than during the midday. This judgement was contrary to that given by five other mothers, who stated that their children's hoarseness was less severe in the morning and more severe at night.

Excessive Vocal Use

The mothers of two subjects, B. F. and B. C., reported excessive crying during infancy in their children. The mothers of four subjects, B. F., J. H., B. M., and M. C., stated that their children were much more talkative and noisy than other children. Seven subjects, B. F., J. H., D. L., B. C., B. M., T. M., and M. C. reportedly yelled excessively while playing with other children out of doors. The individual records revealed that three of the subjects, B. F., T. S., and T. M., were involved in excessive singing prior to the diagnosis of vocal nodules. R. C. stated that he did not use his voice differently from other children. That is, eight of the subjects had a history of vocal abuse.

Statements of Etiology

Of the eight mothers interviewed, four stated that they could think of no factor which may have had a causal relationship with the development of nodules in their children. R. C. stated that he did not know of any reason why he developed vocal nodules. The remaining four mothers did make statements regarding cause. One mother reported that J. H. screams during periods of emotional stress. B. C.'s mother stated that he talks loud to compensate for his small physical size. Another mother said that B. M. is an outgoing child who abuses her voice frequently. Although the specific reasons varied, these three mothers agreed that vocal strain caused the hoarseness and subsequent vocal nodules. However, T. M.'s mother expressed the opinion that the chronic colds led to the development of her child's vocal nodules.

Other Speech Disorders in the Family

Surprisingly, in this sample of nine subjects, seven of the individual records revealed the occurrence of other speech disorders in the families of the subjects. R. C. had a twin brother and sister with articulation disorders. B. F.'s sister and first cousins reportedly had articulation disorders. T. M.'s brother is presently attending speech therapy in the public schools; however, the mother stated that she did not know why the child was enrolled in therapy. J. H. has a brother who "use to stutter." The records on two subjects indicated other incidences of hoarseness in their families. T. S.'s father and B. M.'s mother complained of hoarseness. In summary seven of the subjects' families reportedly had speech disorders. Five of the seven had speech disorders other than hoarseness, and two had other occurrences of hoarseness.

In summary, the individual records yielded information on each subject. This information included a medical history, a description of the onset and development of hoarseness, the variables affecting hoarseness, the mothers' statements of etiology, and a history of vocal use. The individual records also offered information regarding the occurrence of other speech disorders in the families of the subjects. The results indicated that eight of the nine subjects had a history of upper respiratory infections and that four of these eight had these infections during the time when the hoarseness reportedly became noticeable. Surprisingly, only three of the subjects were said to have taken any prescribed medications to control the infections. The age of onset of hoarseness reportedly ranged from three to ten years with six subjects having an onset between the ages of eight and ten years. In describing

the course of development of hoarseness, six of the mothers stated that their children's hoarseness gradually developed from mild to more severe. In addition, several variables were given which were said to have an adverse effect on the hoarseness. These factors included prolonged vocal use such as talking, vocalizing while tense, and excessive singing. When questioned about possible etiology, three of the mothers agreed that vocal strain caused the hoarseness and subsequent vocal nodules. One mother felt that chronic colds led to the development of vocal nodules in her child. Eight subjects had a history of excessive vocal use. Interestingly, the individual records indicated the presence of other speech disorders in the families of seven subjects. Two of these seven families had other occurrences of hoarseness.

Discussion

The results of the airflow evaluations indicated that six subjects had measurable velopharyngeal areas ranging from 1.1mm^2 to 12.2mm^2. For the purpose of this study, velopharyngeal orifice areas of 20mm^2 and greater were considered excessive and indicative of inadequate velopharyngeal closure. This figure was suggested by Warren (1964) as being the area size which separates adequate velopharyngeal closure from inadequate closure. Warren determined this standard measurement in an experiment with ten prosthetically treated cleft palate subjects. In that study, it was determined that nasality in speech becomes apparent to the listener when the orifice area exceeds 20mm^2. Using this standard as a base, all nine subjects in the present study had adequate velopharyngeal closure. Examination of Table 1, page 38, revealed that R. C. had the greatest number of measurable

instances of any of the subjects. Also, during those instances when airflow measurements were made, R. C. had the largest velopharyngeal gaps. However, this subject did manifest an articulation disorder which may have affected the airflow results. That is, while repeating the test phrases, R. C. demonstrated very weak lip closure on the stop consonants [p] and [b] and excessive lateral air leakage on the affricate [s]. This method of articulation may have affected the air pressure in the oropharynx and, therefore, may have affected the airflow measurement of the velopharyngeal port.

Excluding R. C. because of these unusual circumstances, the results on the remaining eight subjects may be viewed with the $20mm^2$ criterion in mind. An obvious conclusion would be that the criterion level may be too large. Since this level was based on the perception of nasality and since the present study was investigating only minimal velopharyngeal inadequacy not necessarily resulting in a perceivable nasal leakage, it is conceivable that the $20mm^2$ level was too generous for the purposes of this study. That is, excessive vocal effort may be necessary long before nasality is perceived in order to achieve acceptable loudness and quality. With this in mind, if the criterion level were lowered by one-fourth and set at $15mm^2$, there still would be no measurements on the remaining eight subjects which even approached this new critical level. If the level were lowered by fifty per cent and set at $10mm^2$, again none of the eight subjects would manifest velopharyngeal gaps larger than the criterion. If the level were lowered by seventy-five per cent and set at $5mm^2$, only one measurement each on D. L. and T. M. in the remaining eight subjects would fall above the critical level. Therefore, it is certain that none of the eight subjects

had inadequate velopharyngeal closure, even with a very stringent criterion.

However, the results do not negate the possibility that these children may have initially utilized excessive effort to achieve this closure. The tension created by this effort may have overflowed throughout the intrinsic muscles of the larynx, thus causing undue strain on the vocal cords and eventually causing vocal nodules. It is possible that such a strain over a prolonged period of time may have served to strengthen the muscles needed to achieve adequate velopharyngeal closure. If such were the case, the subjects would have had residual vocal nodules but good velopharyngeal closure at the time of the airflow evaluation. However, this reasoning would not seem logical when applied to those four subjects who reportedly had upper respiratory infections at the time when hoarseness first became noticeable. Such infections are usually accompanied by edema of the tissue in the nasopharynx and oropharynx. This swelling could only serve to improve velopharyngeal closure. Therefore, it is not likely that these four subjects would have had to exert excessive effort to achieve adequate closure.

Therefore, the proposition that children with vocal nodules also have inadequate velopharyngeal closure, as measured by the airflow procedure, must be rejected. However, there is a possibility that inadequate velopharyngeal closure, as measured by the airflow procedure, may be related to individual children with vocal nodules not used in this study.

For the purpose of this study, an individual record was kept on each subject. This record aided in acquiring information regarding

medical history, onset and development of hoarseness, types of vocal usage, and etiology. In addition, this record was used to note the occurrence of other speech disorders in the families of the subjects.

Upper Respiratory Infections

The individual records indicated that eight of the nine subjects reportedly had a history of upper respiratory infections. This finding tended to support some of the literature on the etiology of vocal nodules. It has been suggested that vocal nodules may be caused by excessive use of the voice while the vocal cords are inflamed. For example, West, Kennedy, and Carr (1947) have stated that repeated misuse of the vocal mechanism and chronic laryngitis or tonsillitis can be related to vocal nodules. Similarly, Alfaro (1960) has credited the development of vocal nodules to vocal abuse or infections or allergic laryngitis.

Medication

In addition to those subjects taking medications for allergic or infectious conditions discussed above, J. H. was said to be taking medication to control hyperactivity. Relating this hyperactivity to vocal usage, the mother of this child recalled that J. H. frequently screams while under tension. This finding appeared to support those who have mentioned excessive vocal use and tension as being related etiologically to the occurrence of hoarseness and vocal nodules. In discussing vocal abuse, Froschels and Jellinek (1958) observed 1000 persons and concluded that contraction of the pharyngeal muscles and 'coup de glotte' were the factors which occurred most often in children speaking under stressful situations. Furthermore, Wilson (1966) has

recommended relaxation of the laryngeal muscles in the treatment of children who have been diagnosed as having vocal nodules as a result of excessive tension.

Age of the Onset of Hoarseness

Six of the subjects reportedly developed hoarse voices between the ages of eight and ten years, and one subject's voice became noticeably hoarse at the age of six. Only two of the subjects were said to have developed hoarseness at a preschool age. Therefore, the results indicated that, in these children, hoarseness did not become noticeable until the children reached school age. One possible explanation for the development of hoarseness during the school years may be that children of school age usually have a greater opportunity to participate in activities where vocal abuse may occur.

Development of Hoarseness

Six mothers recalled that their children's hoarseness developed gradually from slight to more severe. These reports appeared to reflect the literature's description of the gradual process involved in the development of vocal nodules. That is, as the nodule advances from a soft epithelial lesion to a hard growth composed of thickened epithelium, the degree to which the nodule interferes with the approximation of the vocal cords may increase. Therefore, a gradual increase in hoarseness would seem to reflect the gradual nature of the development of vocal nodules.

Variables Affecting Hoarseness

The individual records on eight subjects indicated that prolonged talking and vocal abuse negatively affected hoarseness, a result which supports the findings of Wilson (1966). R. C. failed to report such variation. The records on two subjects, B. F. and J. H. stated that emotional tension adversely affected the hoarseness, supporting the possibility that tension may be a cause for vocal nodules (Arnold, 1962).

Excessive Vocal Use

Eight of the nine subjects had a history of excessive vocal use. This excessive use took the form of excessive crying in infancy, talking, yelling, and singing. Although highly subjective, this information did seem to reinforce the concept of vocal abuse as a cause of vocal nodules. R. C., the subject who served as his own informant, was the only subject who did not report a history of excessive vocal use.

Statements of Etiology

Three mothers expressed the opinion that vocal strain was the cause of their children's vocal nodules. This opinion is strongly supported by the literature. Greene (1957, p. 78) has referred to this vocal strain as the ". . . individual's habitual method of forcing the voice." Arnold (1962, p. 214) has called the factor ". . . excessive and uncontrolled vocal expression." Wilson (1967, p. 19) has written about ". . . excessive strained phonation during play. . . ." In addition, one mother attributed the cause of the nodules to chronic colds and laryngitis. Therefore, this study has found additional support for the opinion that vocal strain and upper respiratory infections are causative factors in the development of vocal nodules.

Other Speech Disorders in the Families

Seven of the nine subjects reportedly had other members of their families who had speech disorders. It was difficult to account for this relatively high percentage in a sample of nine. No information was available on the nature of the diagnosis or treatment of the speech disorders in five of the families. The records did reveal that the two parents who complained with hoarseness themselves had not sought medical treatment or speech therapy for the hoarseness. The explanation of a multiple occurrence of hoarseness in a family would seem to be dependent on various theories of etiology. It could well be that vocal habits among family members are similar. Therefore, if vocal abuse were the cause of hoarseness, then it is highly conceivable that a child could learn poor vocal habits from a parent. Moreover, there could be physiological factors involved. As Rubin and Lehrhoff (1962) have suggested, perhaps vocal abuse is relative to the speaker. That is, what constitutes vocal abuse for one speaker may not necessarily be detrimental to another speaker. Therefore, a weakness in the parent's vocal mechanism may be inherited by the child. Another explanation may lie in the possibility of multiple occurrences of upper respiratory infections in the same family. Also, the presence of hoarseness in a child and parent may possibly be unrelated etiologically.

In summary, the airflow procedure failed to reveal inadequate velopharyngeal closure in any of the subjects. In fact, the compiled information from the individual records appeared to confirm the theories set forth in the literature on the etiology of vocal nodules. That is, the high incidence of upper respiratory infections and vocal abuse in the sample supported the theory that poor vocal habits, particularly

during a period when the vocal cords are inflamed, can lead to hoarseness and subsequently to vocal nodules. In addition, two subjects reporting a history of tension supported the theory that tension can be related to vocal abuse. The multiple occurrence of hoarseness in the same family could be explained as being due to similar etiological factors such as poor vocal habits, inherently weak vocal mechanisms, or chronic upper respiratory infections.

CHAPTER V

SUMMARY AND CONCLUSIONS

The etiology of vocal nodules is of interest to speech pathologists. However, the literature reporting the incidence and etiology of vocal nodules is inconclusive. McWilliams, Bluestone, and Musgrave (1969) have suggested that the high frequency of vocal nodules in children with congenital clefts of the palate may be related etiologically to velopharyngeal inadequacy in these children. Therefore, the present study was conducted to determine if non-cleft palate children with vocal nodules also manifest minimal velopharyngeal inadequacy. The procedure for the study involved conducting airflow evaluations on nine children, ranging in age from eight to twelve years, with vocal nodules. In addition, an individual record was compiled on each subject's medical history, the onset and development of hoarseness, the management of the vocal nodules, the variables affecting hoarseness, and a history of vocal use. An additional section provided space to record the occurrence of other speech disorders in the subject's family.

The results of the study revealed that all nine subjects had adequate velopharyngeal closure during speech as measured by the airflow procedure. Therefore, the hypothesis that these non-cleft palate children with vocal nodules also have velopharyngeal inadequacy must be rejected. The individual record results supported the literature in suggesting that vocal abuse, particularly during a time when the vocal cords are inflamed, is related etiologically to vocal nodules. The

results on two subjects supported the theory that tension may be related to vocal abuse.

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APPENDIX I

LETTER OF INTRODUCTION

Date

Dear

A study is being conducted at the University of North Carolina at Greensboro in an attempt to better understand the causes of vocal nodules. As your child previously obtained services at (the Speech and Hearing Center, the Health Department, from Dr. L. L. Patseavouras), your names are listed in (our, their, his) files. Therefore, we are contacting you in the hope that you and your child would be willing to participate.

The study is designed to measure how far the soft palate raises toward the back of the throat during speech. We will contact you by telephone in a few days to tell you more about the extent of your participation and to answer any questions you may have.

It is our hope that this study may benefit children with vocal nodules. You and your child's participation would be an extremely helpful contribution and would be sincerely appreciated.

Very truly yours,

Bonnie W. Amos

Mariana Newton, Ph. D.
Assistant Professor of
Speech Pathology

APPENDIX II

STATEMENT OF INFORMED CONSENT

We would like you and your child to participate in a research project which is designed to measure how much a person's soft palate raises toward the back of the throat during speech. We would like for you to come to the University of North Carolina at Greensboro Speech and Hearing Center for two days. On one day we will take your child to the Dental Research Center in Chapel Hill, North Carolina. On that day, we will conduct an airflow procedure on your child. On the second day, we will ask you questions about your child and conduct a hearing evaluation. The hearing and airflow examinations will be conducted by or under the supervision of a licensed clinician who does these procedures as a clinically acceptable part of his practice.

The hearing and airflow evaluations are safe and are currently in clinical use in the United States by certified clinicians. There are no side effects.

The speech samples, which are necessary for the airflow procedure, will be recorded for our records. These tapes will be considered confidential, and you and your child's identity will not be revealed.

The above statement has been read aloud to me. I understand the nature and extent of my participation and my child's participation in the study, and by my signature I consent to participate as outlined above.

_____
Signature

_____
Bonnie W. Amos

APPENDIX III

INDIVIDUAL RECORD

I. IDENTIFICATION

Name: _____ Sex: _____
Age: _____ Date: _____
Birthdate: _____ Referred by: _____
Address: _____ Examiner: _____
Parents: _____ Telephone: _____

II. MEDICAL HISTORY

Chronic Colds: _____ Chronic sinusitis: _____
Scarlet fever: _____ Pneumonia: _____
Diptheria: _____ Rheumatic fever: _____
Whooping cough: _____ Influenza: _____
Allergies: _____ Tonsillitis: _____
Frequent sore throats: _____
What is your child's present state of health? _____
_____
Is your child on any medication? _____
_____

III. INFORMATION CONCERNING NODULES

Bilateral: _____ Other Laryngeal anomalies:
Unilateral: _____ _____
Treatment: Surgery: _____
_____ Date: _____
Physician: _____
Therapy: _____ Dates: _____
Emphasis during therapy: _____
_____
Therapist: _____

IV. VOCAL HISTORY

When and under what circumstances did you first notice your child's voice problem?

Did the voice problem come on gradually or suddenly?

What do you think caused the problem?

Has the problem decreased or increased since it began?
Describe:

What time of the day is your child's voice best? Worse?

Does the problem vary with the season of the year or the weather?

Does the problem vary with the amount of talking that your child does?

Does the problem vary with any other conditions?

Is your child's voice similar to some other person's in your family? Whose?

Do you know of any speech disorders in your family?

Amount and kind of vocal usage: As an infant, did your child cry or scream a lot? _____ Is your child very talkative or noisy? _____ Does your child yell excessively on the playground or in competitive sports? _____ Does your child sing in a choir? _____ What activities does your child participate in which involve loud or continuous talking? _____

V. HEARING

Results of pure-tone screening test: